Banchereau Jacques; Rousset Francoise
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JOURNAL: Journal of Immunology 153 (3):p1333-1339 1994
ISSN: 0022-1767
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RECORD TYPE: Abstract
LANGUAGE: English

6/3/45 (Item 3 from file: 73)
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04764451 EMBASE No: 1991257805
Experimental autoimmune oophoritis. II. Both lymphoid cells and antibodies are successful in adoptive transfer
Damjanovic M.
Immunology Research Center, Vojvode Stepe 458, 11221 Belgrade Yugoslavia
Autoimmunity (AUTOIMMUNITY) (United Kingdom) 1991, 9/3 (217-223)
CODEN: AUIME ISSN: 0891-6934
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH

6/3/46 (Item 4 from file: 73)
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04216077 EMBASE No: 1990098619
Autoimmune orchitis and oophoritis
Tung K.S.K.; Mahi-Brown C.A.
Washington University School of Medicine, Box 8118, 660 South Euclid Avenue, St. Louis, MO 63110 United States
Immunology and Allergy Clinics of North America (IMMUNOL. ALLERGY CLIN. NORTH AM.) (United States) 1990, 10/1 (199-214+ix)
CODEN: INCAE ISSN: 0889-8561
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

6/3/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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09885688 BIOSIS NO.: 199598340606
Contribution of CD28/CTLA4/B7 and gp39/CD40 costimulation pathways in clonal expansion and functional acquisition of self reactive T cells.
AUTHOR: Griggs Nathan(a); Agersborg Sally; Noelle Randolf; Ledbetter Jeffrey; Linsley Peter; Tung Kenneth
AUTHOR ADDRESS: (a)Dep. Pathol., Univ. Virginia, Charlottesville, VA 22908 **USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (21A):p141 1995
CONFERENCE/MEETING: Keystone Symposium on Control and Manipulation of the Immune Response Taos, New Mexico, USA March 16-22, 1995
ISSN: 0733-1959
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Banchereau Jacques; Rousset Francoise
AUTHOR ADDRESS: INSERM U346, Hopital Edouard Herriot, 69467 Lyon Cedex 03 **France
JOURNAL: Journal of Immunology 153 (3):p1333-1339 1994
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

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04764451 EMBASE No: 1991257805
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Autoimmunity (AUTOIMMUNITY) (United Kingdom) 1991, 9/3 (217-223)
CODEN: AUIME ISSN: 0891-6934
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH

6/3/46 (Item 4 from file: 73)
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Autoimmune orchitis and oophoritis
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Washington University School of Medicine, Box 8118, 660 South Euclid Avenue, St. Louis, MO 63110 United States
Immunology and Allergy Clinics of North America (IMMUNOL. ALLERGY CLIN. NORTH AM.) (United States) 1990, 10/1 (199-214+ix)
CODEN: INCAE ISSN: 0889-8561
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

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DIALOG(R)File 5:Biosis Previews(R)
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09885688 BIOSIS NO.: 199598340606
Contribution of CD28/CTLA4/B7 and gp39/CD40 costimulation pathways in clonal expansion and functional acquisition of self reactive T cells.
AUTHOR: Griggs Nathan(a); Agersborg Sally; Noelle Randolf; Ledbetter Jeffrey; Linsley Peter; Tung Kenneth
AUTHOR ADDRESS: (a)Dep. Pathol., Univ. Virginia, Charlottesville, VA 22908 **USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (21A):p141 1995
CONFERENCE/MEETING: Keystone Symposium on Control and Manipulation of the Immune Response Taos, New Mexico, USA March 16-22, 1995
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

6/3/35 (Item 35 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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Set     Items   Description
S1         35   (OOPHORITIS OR THYROIDITIS) AND (GP39 OR CD40L OR CD40(W)L-
               IGAND)
S2         19   RD S1 (unique items)
S3        588   (OOPHORITIS OR THYROIDITIS) (20N) (AUTOIMMUN?) AND REVIEW?
S4         17   S3 AND PY=1993
S5         13   RD S4 (unique items)
? s s3 and (inhibit? or suppress? or prevent? or block?) (10n)(antibod?)

Processing
Processing
             588  S3
         3436985  INHIBIT?
          686427  SUPPRESS?
         1755019  PREVENT?
         1082733  BLOCK?
         1687525  ANTIBOD?
          182149  (((INHIBIT? OR SUPPRESS?) OR PREVENT?) OR
                  BLOCK?)(10N)ANTIBOD?
      S6      15  S3 AND (INHIBIT? OR SUPPRESS? OR PREVENT? OR BLOCK?)
                  (10N)(ANTIBOD?)
? rd s6

...completed examining records
      S7      15  RD S6 (unique items)
? t s7/7/all


 7/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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03331552   BIOSIS NO.: 000072059656
IDIOTYPIC NETWORKS AND THEIR POSSIBLE EXPLOITATION FOR MANIPULATION OF THE
  IMMUNE RESPONSE
AUTHOR: ROITT I M; MALE D K; GUARNOTTA G; DE CARVALHO L P; COOKE A; HAY F C
  ; LYDYARD P M; THANAVALA Y; IVANYI J
AUTHOR ADDRESS: DEP. IMMUNOL., MIDDLESEX HOSP. MED. SCH., LONDON W1.
JOURNAL: LANCET 1 (8228). 1981. 1041-1045. 1981
FULL JOURNAL NAME: Lancet
CODEN: LANCA
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: The theory of immune response regulation by an
  idiotype-anti-idiotype network involving helper and suppressor T cells
  bearing idiotype and anti-idiotype receptors is reviewed.
  Antigen-mediated immunoregulation is also discussed. The potential for
  treating such human autoimmune diseases as Hashimoto's
  thyroiditis and rheumatoid arthritis by intervening in the idiotype
  network regulatory chain is discussed. A possible approach, involving
  injection with specific anti-idiotype antibodies to generate in
  vivo antigen-specific suppressor T cells, is proposed.


 7/7/2     (Item 1 from file: 73)
DIALOG(R)File  73:EMBASE
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11082900     EMBASE No: 2001102076
  Chronic urticaria and Hashimoto's thyroiditis: Six case reports
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URTICAIRE CHRONIQUE ET THYROIDITE DE HASHIMOTOHASHIMOTO : PROPOS DE SIX CAS

Delevaux I.; Andre M.; Tridon A.; Aumaitre O.
O. Aumaitre, Service de medecine interne, CHU, Hopital Gabriel-Montpied, BP 69, 63003 Clermont-Ferrand cedex 1 France
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Revue de Medecine Interne (REV. MED. INTERNE) (France) 2001, 22/3 (232-237)
CODEN: RMEID ISSN: 0248-8663
DOCUMENT TYPE: Journal ; Article
LANGUAGE: FRENCH SUMMARY LANGUAGE: ENGLISH; FRENCH
NUMBER OF REFERENCES: 22

Purpose. - Chronic urticaria is a common skin disorder. The cause is rarely determined. **Autoimmune** diseases, particularly **autoimmune thyroiditis**, have been implicated in the occurrence of chronic urticaria. Methods. - We **reviewed** clinical records of patients with Hashimoto's disease and chronic unicaria. Results. - In our department, six patients had presented chronic urticaria associated with Hashimoto's thyroiditis: four patients, of which three treated with L-thyroxine were euthyroid, the other two were hypothyroid. Hashimoto's thyroiditis had been diagnosed for three patients during the investigation of chronic urticaria. Three patients developed chronic urticaria though they were treated with thyroid suppression for Hashimoto's disease. Two of them had a dramatic improvement with opotherapy. One patient who was euthyroid without treatment improved with hormonal therapy. The fourth patient had a partial remission with thyroid hormones and was cured with corticotherapy. Conclusion. - The mecanism by which thyroid autoimmunity is associated with urticaria is poorly understood. A cross-linking of IgE receptors of mastocytes induced by antithyroid antibodies may be a cause of histamine release. Hormonal therapy may be a potent event for the clinical improvement by the **suppression** of chronic thyroid stimulation. Assay of thyroid hormone and antithyroid **antibodies** should be performed in patients with chronic urticaria. Discovery of Hashimoto's thyroiditis with chronic urticaria requires thyroid hormone replacement not only in hypothyroid but also euthyroid patients. (c) 2001 Editions scientifiques et medicales Elsevier SAS.

7/7/3 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

07240141 EMBASE No: 1998138991

Interluekin-1beta induced transient diabetes mellitus in rats: A model of the initial events in the pathogenesis of insulin-dependent diabetes mellitus?

Reimers J.I.
J.I. Reimers, Aeblekrogen 12, DK-2830 Virum Denmark

Danish Medical Bulletin (DAN. MED. BULL.) (Denmark) 1998, 45/2 (157-180)
CODEN: DMBUA ISSN: 0907-8916
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 243

When aiming at preventing IDDM in man, knowledge of the molecular mechanisms leading to beta cell destruction may facilitate identification of new possible intervention modalities. A model of IDDM pathogenesis in man suggests that cytokines, and IL-1 in particular, are of major importance in the initial events (Nerup et al 1994) (Fig. 1). In vitro rat experiments demonstrated that rhIL-1beta inhibits beta cell function and induces beta cell death both in isolated islets of Langerhans and in the isolated perfused pancreatic gland. With the long term goal of identifying

new modalities capable of preventing IDDM in man, the aim of this **review** was to investigate the effects of rhIL-1beta on beta-cell function and viability in normal rats. This **review** discussed 1) the pharmacokinetics of IL-1beta in rats as the basis for choice of route of administration and dose of rhIL-1beta 2) the effects and molecular mechanisms of IL-1beta on temperature and food intake use as control parameters for successful injection of rhIL-1beta in rats, 3) the effects of one or more injection of IL-1beta induced beta cell inhibition in vivo, and some possible intervention modalities based on the molecular mechanisms, 5) the effects of IL-1beta on spontaneous diabetes mellitus in DP BB rats, and 6) the effects of IL-1beta on rat beta cell function. Finally, this **review** discussed the effects of IL-1beta on human beta cells in vitro, and the clinical relevance of these experiments, with spectral reference to a clinical trial with the aim of preventing IDDM in man. The pharmacokinet studies suggested the IL-1beta is distributed according to a two-compartment model with a first-order elimination. Interleukin-1beta reached all the investigated organs in the rats, was accumulated in kidneys and was excreted in the urine. The data suggested that IL-1beta also accumulated in the islets of Langerhans. After injection of 4.0 mug/kg pathophysiologically relevant concentrations of rhIL-beta were reached and intact rhIL-1beta persisted for up to 5 hrs in plasma. Peripheral injections of IL-1beta dose dependently induced fever and anorexia in rats, probably via induction of PGEinf 2 in the beam or in peripheral tissues thereafter passing the blood-brain barrier. Nitrix oxide produced by cNOs seems to be a molecular mediator of IL-1beta induced fever but not of anorexia. Fever and anorexia are well described effects of IL-1beta in rats, and are as such useful control parameters of the absorption and biological activity of IL-1beta after peripheral injection. Injections of rhIL- 1beta to normal, non-diabetes one rats induced initial beta cell stimulation followed by inhibition in accordance with in vitrodata. Furthermore, induction of peripheral insulin resistance coincided with beta cell inhibition after one daily injection for 5 days, leading to a transient diabetes mellitus like state characterized by hyperglycemia and hypoinsulinemia. At this time point, electron-microscopy did not demonstrate beta cell destruction. However, IL-1beta induced intercellularly edema and microvillous processes on the beta cells, which might be early evidence of apoptosis. The diabetes mellitus-like state was not aggravated if the daily injections were continued beyond 5 days. Daily injections of rhIL-1beta for 2 to 4 weeks induced formation of **blocking** IL-1beta-**antibodies** in normal rats. Hence, injections exceeding 2 weeks should only be performed using species homologous IL-1beta. The molecular mechanism of IL-1beta induced beta cell inhibition in rats in vivo as in vitro, are likely to involve binding of IL- 1beta to the IL-1RtI, since the IL-1RtII is considered to be a decor reactor vivo as in vitro, suggesting that differences in sensitivity on alpha and beta cells in vivo should be due to differences in the receptor occupancy needed to trigger the intracellular events, or differences in number and type of IL-1R's on alpha and beta cells. Based on data which demonstrated that inhibition of iNOS prevented the IL-1beta induced diabetes mellitus-like state in rats, and that iNOS protein content in islets was higher in rats sensitive to IL-1beta induced diabetes mellitus-like state in vivo compared to non- sensitive rats, it was suggested that the post-receptor events involve induction of NO via iNOS. Induction of FOR has been suggested to be of importance in the deleterious effects of IL-1beta on beta cells in vitro. iNOs inhibition completely prevented the IL-1beta induced diabetes mellitus like state, which suggested that induction of FOR is not of major importance in vivo. The IL-1beta induced diabetes mellitus-like state in rats was suggested to be a model of the initiating phase in the pathogenes of IDDM. Studies using this model for investigation of new possible intervention modalities demonstrated that reduced beta cell activity due to fasting, inhibited the IL-1beta induced diabetes millitus-like state. This is an correspondence with the 'moving target' hypothesis suggesting that the sensitivity of the beta cells to IL-1beta depends on the functional activity of the cells. Administration of the synthetic immune-stimulator

Linomide, known to increase natural killer cell, T cell, and B cell activity, did not prevent the IL-1beta induced diabetes mellitus in rats. However, this was not unexpected, since the initiating events in the pathogenesis of IDDM are hypothesised to involve only macrophages, and not natural killer cell, T cell, or B cells. Furthermore, an increased glucose intake or co-injection of the IFNalpha inducer Poly I C did not aggravate IL-1beta induced diabetes mellitus. In studies on DP BB rats, administration of high dose IL-1beta accelerates the onset but does not have any effect on the onset but does not have any effect on the incidence of diabetes mellitus, whereas low dose IL-1beta reduced the incidence, when compared to an ad libitum fed control group. Interleuking- 1beta injections induced increased blood glucose concentration before and at onset of diabetes mellitus in DP BB rats, which was suggested to be due to the combination of rhIL-1beta induced peripheral insulin resistance and inhibition of beta cell function. Bolus injections of IL-1beta or other cytokines can not mimic increased local concentration of the cytokine in the islet. Hence, the importance of IL-1beta and other cytokines in the pathogenesis of diabetes mellitus in the DP BB rats could be investigated by expressing the cytokines, cytokine receptors, or receptor antagonists in the islets of Langerhans, or by using the so called 'knock-out technique'. Interleukin-1beta inhibits thyroid epithelial cell function both in vivo as in vitro. However, the data suggest that IL-1beta does not inhibit thyroid epithelial cell function via induction of NO, which is in contrast to beta cells. Observations further suggest that the resistance of the thyroid epithelial cells to IL-1beta induced cell death is due to a low inducility of iNOS and low NO production in thyroid epithelial cells. The pathogenesis of **autoimmune thyroiditis** has been proposed to consist of two phases, an initiating phase and an amplification phase, which is in correspondence to the proposed model for the pathogenesis of IDDM. However, it is proposed that specific anti-bodies initiates the process in the pathogenesis of **autoimmune thyroiditis**, whereas antibodies are only described as epiphenomenons in the pathogenesis of IDDM. Furthermore, IL-1beta aggravated the spontaneous **thyroiditis** and induced hypothyroidism in DP BB rats, without any effect on diabetes incidence of insulitis. This also supports that pathogeneses of **autoimmune thyroiditis** and IDDM are different. The molecular mechanisms leading to aggravation of the **thyroiditis** and hypothyroidism in DP BB rats needs further investigation. Most data suggest that human beta cells are more resistant to IL-1beta that rat beta cells in vitro. This is likely to be due to genetically determined differences in expression of protective stress proteins amide could prevent spontaneous and chemically-induced diabetes mellitus in rodents, led to experiments demonstrate that nicotamide prevents cytokine induced inhibition of human beta cells in vitro, and to a large- scale multinational, randomized, double-blind, prospective, placebo- controlled trial investigating the potential effect of nicotinamide in the prevention of IDDM in high risk individuals. It is concluded that the result of IL-1beta injections in vivo in rats (beta cell inhibition and hyperglycemia) are in agreement with the in vitro studies. The in vivo data support the Copenhagen Model of IDDM Pathogenesis (Fig. 1), suggesting that the initiating events in the pathogenetic process leading to beta cell inhibition and destruction, could be due to IL-1beta alone or in combination with TNFalpha. However, beta cell destruction was not demonstrated after peripheral injection of IL-1beta, suggesting that the intra-islet concentration of IL-1beta reached was not sufficiently high or the exposure time was to short to induce beta cell destruction. Further investigation, as described previously, is needed to clarify if continuously high intra-islet concentrations of IL-1beta can induce beta cells destruction in rats. Using the rat model of the initial events in the IDDM pathogenesis for screening of possible intervention modalities, demonstrated that inhibition of iNOS induced NO production prevented the IL-1beta induce diabetes mellitus like condition in vivo. The potential effect of iNOS inhibitors should be considered for investigation in prediabetic man. Treatment with the aim of preventing onset of IDDM is likely to be life-long. Thus, potential intervention modalities should be

without adverse effects.

7/7/4 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

04124275 EMBASE No: 1990006653
Humoral and cellular reactions in **autoimmune thyroiditis**
HUMORALE AND ZELLULARE FAKTOREN BEI DER IMMUNTHYREOIDITIS
Bogner U.; Schleusener H.
Endokrinologische Abteilung, Klinikum Steglitz, Medizinische Klinik, Hindenburgdamm 30, D-1000 Berlin 65 Germany.
Aktuelle Endokrinologie und Stoffwechsel (AKTUEL. ENDOKRINOL. STOFFWECHSEL) (Germany) 1989, 10/SUPPL. 2 (140-145)
CODEN: AENSD ISSN: 0172-4606
DOCUMENT TYPE: Journal; Review
LANGUAGE: GERMAN SUMMARY LANGUAGE: ENGLISH

Microsomal and thyroglobulin antibodies are detectable in a high frequency in **autoimmune thyroiditis**. The microsomal antigen could be identified as the thyroid peroxidase. TSH-receptor antibodies are only present in a minor frequency in **autoimmune thyroiditis** and seems to be present in a higher incidence in primary myxoedema. Their role of the induction of neonatal hypothyroidism by transplacental passage from the mother is described. Although not characterized in further detail antibodies against the 'second colloid antigen' are frequently detectable in **autoimmune thyroiditis**. Antibody-dependent complement-mediated cytotoxicity against thyrocytes and other target cells are found by some investigators and showed in most cases a good correlation to the titres of the microsomal antibodies. The analysis of the intrathyroidal lymphocyte subsets showed a predominance of the CD8 (**Suppressor**/cytotoxic) T cells. Cytotoxic **antibodies** determined by an **antibody**-dependent cell-mediated cytotoxicity assay against human thyroid cells were detectable in about 70% of patients with HT. These antibodies showed no correlation to other antibodies measurable in HT. This finding implies that cytotoxic antibodies are either directed against an unknown antigen or binds to an epitope of the peroxidase molecule which is not detectable by the conventional test for microsomal antibodies. Dependent on the methods controversial results are described for the direct (NK cell) mediated cytotoxicity in HT.

7/7/5 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

03638649 EMBASE No: 1988088085
Immunologically mediated hypothyroidism
Dussault J.H.; Rousseau F.
Department of Medicine, Centre Hospitalier de l'Universite Laval, Sainte-Foy, Que. Canada
Endocrinology and Metabolism Clinics of North America (ENDOCRINOL. METAB. CLIN. NORTH AM.) (United States) 1987, 16/2 (417-429)
CODEN: ECNAE ISSN: 0889-8529
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

In this article, new avenues in the understanding of immunologic processes involved in hypothyroidism have been explored. The discovery of a family of TSH-R directed **antibodies**, including TSI-**block**, TGI, and TGI-**block**, has afforded perspectives on the etiology of autoimmune thyroid disorders. Thus, whereas TSI and TSI-block influence thyroid function, TGI and TGI-block are involved in thyroid cell

proliferation and maturation. We have focused on three clinical entities that have been elucidated relatively recently - namely, silent thyroiditis, postpartum thyroiditis, and congenital hypothyroidism. Silent thyroiditis, a common form of transient thyroiditis, yields very few clinical symptoms or signs but significant alterations in biological tests, including a thyroid sup 1sup 3sup 1I uptake compatible with silent thyroid destruction. Although an **autoimmune** etiology is really not certain at this moment, it is not completely excluded. Silent **thyroiditis** does not usually require therapy, except in the rare cases in which symptoms are very severe. Postpartum thyroiditis, probably a special form of silent lymphocytic thyroiditis, differs from silent thyroiditis only by its relation to pregnancy and its higher rate of persistent thyroid disease. It has a high prevalence in pregnant women (5.5 to 10.2 per cent) in all populations studied, and may be responsible for a substantial proportion of cases of postpartum depression. Although the etiology is not clear, an autoimmune process seems to be involved. Although prediction of this state is difficult, a previous episode or high titers of microsomal antibodies in the first trimester show good correlations with the disease. Thyroid hormone replacement therapy is recommended for persistent disease. Congenital hypothyroidism appears to be mediated by passively transferred maternal **blocking antibodies**. TSI-**block** is likely responsible for the transient form of congenital hypothyroidism in the same way that TSI may cause transient congenital thyrotoxicosis. Passive transfer of maternal TGI-block appears to be causal in the majority of newborns with the sporadic form of congenital hypothyroidism. Early (in utero) onset of the disease could explain why 15 per cent of adequately treated infants subsequently demonstrate subtle neurologic sequelae. Because screening procedures for TGI-**blocking antibodies** are being made available, it should be possible to detect those potentially severe in utero cases and commence thyroid hormone replacement therapy before birth.

7/7/6 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

02970665 EMBASE No: 1985064625
Autoimmunity of thyroid disease with emphasis on 'Graves' disease
Van Ouwerkerk B.M.; Krenning E.P.; Docter R.; et al.
Department of Internal Medicine and Clinical Endocrinology, Hospital 'Dijkzigt', Erasmus University, 3015 GD Rotterdam Netherlands
Netherlands Journal of Medicine (NETH. J. MED.) (Netherlands) 1985, 28/1 (32-39)
CODEN: NLJMA
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH

Graves' disease (GD), Hashimoto's **thyroiditis** (HT) and primary myxoedema should be regarded as thyroid **autoimmune** diseases. GD is estimated to occur in 0.4 per cent of caucasians and is due to autoantibodies that stimulate the thyroid follicular cell, causing hyperthyroidism and often diffuse goitre. GD occurs about 10 times as often in women as in men. HT and primary myxoedema are found four times as often in women as in men and are characterized by antibodies against thyroglobulin (in 2-15 per cent of all women between 25 and 75 years old) and microsomes (in 7 per cent of all adults), leading to failure of thyroid hormone production. Recently it has been suggested that thyroid **antibodies** which **inhibit** the action of thyrotropin may also play a role in the development of primary hypothyroidism. The purpose of this **review** is to describe and discuss the different pathogenetic aspects of autoimmune thyroid disease.

7/7/7 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

02529933 EMBASE No: 1983003944
Autoimmunity and hypothyroidism
Dessaint J.P.; Wemeau J.L.
Serv. Immunol., UER Med., Univ. Lille, F-59000 Lille France
Hormone Research (HORM. RES.) (Switzerland) 1982, 16/5 (329-337)
CODEN: HRMRA
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH

Primary myxedema and hypothyroid Hashimoto's disease provide a well-documented example of organ-specific autoimmunity in man. Very slight modifications or increased release of thyroglobulin or thyroid antigens in the circulation may cause the rupture of autotolerance for the normal thyroid components, at least when individuals have a genetic predisposition to **autoimmune thyroiditis** (possibly associated with a predisposition to other **autoimmune** diseases). The demonstration of an association between HLA and **thyroiditis**, however, requires additional studies. The basic immunological abnormality responsible for **autoimmunization** against thyroid components is a defect in suppressor T cells, shown in experimental animals but not firmly established in man. The results of autoimmunization will be the appearance of cytotoxic mechanisms that lead to destruction of the thyroid follicle with progressive fibrosis, antibody-dependent cell-mediated cytotoxicity apparently being of major importance. A recent report shows, in addition, that thyroid atrophy in primary hypothyroidism is associated with the production of **antibodies** that **block** the thyroid-growth-promoting activity of TSH. The recent progress made in our understanding of **autoimmune thyroiditis** will certainly contribute to improving our knowledge of how and when **autoimmunization** might develop in man.

7/7/8 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

01159634 EMBASE No: 1978290425
Autoimmune thyroid diseases: Graves' and Hashimoto's
Solomon D.H.; Beall G.N.; Terasaki P.I.; et al.
Dept. Med., UCLA Sch. Med., Los Angeles, Calif. United States
Annals of Internal Medicine (ANN. INTERN. MED.) (United States) 1978, 88/3 (379-391)
CODEN: AIMEA
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH

Thyroid-related **autoimmune** diseases (Graves' thyroid disease, Graves' ophthalmopathy, and Hashimoto's **thyroiditis**) may occur alone or in any association. The diagnosis of Hashimoto's thyroiditis requires multiple criteria; pathologic changes in the thyroid are not due to **antibodies** but may result from cytotoxic lymphocytes or a deficiency of **suppressive** T cells. In Graves' and Hashimoto's diseases the increased prevalence of HLA-B8 may not be significant, but that of HLA-AW30 in Hashimoto's disease is. In 48 first-degree relatives of patients with Graves' disease, thyroid abnormalities were frequent but not correlated with HLA type. Elevated serum thyroglobulin levels in all patients with hyperthyroidism fell to normal after surgical resection or radioiodine therapy. Patients whose illness recurred after antithyroid drug treatment was stopped had higher pretreatment thyroglobulin levels and no fall during treatment; those whose illness remitted had lower initial levels and a

significant fall during treatment. Sodium ipodate lowered serum triidothyronine and thyroxine levels in hyperthyroid patients and may be useful in the treatment of hyperthyroidism.

7/7/9 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE

00535027 EMBASE No: 1976090588
Cell mediated immunity and immune complexes in thyroid disease
Calder E.A.; Irvine W.J.
Dept. Therapeut., Roy. Infirm., Edinburgh United Kingdom
Clinics in Endocrinology and Metabolism (CLIN. ENDOCRINOL. METAB.)
1975, 4/2 (287-318)
CODEN: CEDMB
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH

In the late 1950s and early 1960s interest in the immunological aspects of thyroid disease was centred around the identification and possible pathogenic significance of the thyroid specific autoantibodies present in the serum of patients with autoimmune thyroid disease. It soon became apparent, however, that humoral antibody alone was not solely responsible for the production of thyroid cell damage in vivo. The invariable presence of leucocytes of the histiocyte and lymphocyte series in the inflammatory lesions of patients with autoimmune thyroid disease suggested that cell mediated hypersensitivity reactions might play some role in its production. Techniques designed to be in vitro correlates of cell mediated immunity (CMI) have subsequently been developed and used to study this phenomenon in a variety of disease states. The purpose of this chapter is to **review** the evidence which exists, based on the use of such in vitro tests, to implicate cell mediated (CMI) have subsequently been developed and used to study this phenomenon in a variety of disease states. The purpose of this chapter is to **review** the evidence which exists, based on the use of such in vitro tests, to implicate cell mediated immune mechanisms in the pathogenesis of autoimmune thyroid disease.

7/7/10 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11334342 21170595 PMID: 11270265
[Chronic urticaria and Hashimoto-Hashimoto's thyroiditis: report of 6 cases]
Urticaire chronique et thyroidite de HashimotoHashimoto: a propos de six cas.
Delevaux I; Andre M; Tridon A; Aumaitre O
Service de medecine interne, CHU, hopital Gabriel-Montpied, BP 69, 63003 Clermont-Ferrand, France.
La Revue de medecine interne (France) Mar 2001, 22 (3) p232-7, ISSN 0248-8663 Journal Code: SGJ
Languages: FRENCH
Document type: Journal Article ; English Abstract
Record type: Completed
PURPOSE: Chronic urticaria is a common skin disorder. The cause is rarely determined. **Autoimmune** diseases, particularly **autoimmune thyroiditis** , have been implicated in the occurrence of chronic urticaria. METHODS: We **reviewed** clinical records of patients with Hashimoto's disease and chronic urticaria. RESULTS: In our department, six patients had presented chronic urticaria associated with Hashimoto's thyroiditis: four patients, of which three treated with L-thyroxine were euthyroid, the other two were hypothyroid. Hashimoto's thyroiditis had been diagnosed for three patients during the investigation of chronic urticaria.

Three patients developed chronic urticaria though they were treated with thyroid suppression for Hashimoto's disease. Two of them had a dramatic improvement with opotherapy. One patient who was euthyroid without treatment improved with hormonal therapy. The fourth patient had a partial remission with thyroid hormones and was cured with corticotherapy. CONCLUSION: The mechanism by which thyroid autoimmunity is associated with urticaria is poorly understood. A cross-linking of IgE receptors of mastocytes induced by antithyroid antibodies may be a cause of histamine release. Hormonal therapy may be a potent event for the clinical improvement by the **suppression** of chronic thyroid stimulation. Assay of thyroid hormone and antithyroid **antibodies** should be performed in patients with chronic urticaria. Discovery of Hashimoto's thyroiditis with chronic urticaria requires thyroid hormone replacement not only in hypothyroid but also euthyroid patients.
Record Date Created: 20010328

7/7/11 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10693834 20325122 PMID: 10865094
Encephalopathy associated to autoimmune thyroid disease: a more appropriate term for an underestimated condition?
Canton A; de Fabregas O; Tintore M; Mesa J; Codina A; Simo R
Department of Endocrinology, Hospital Vall d'Hebron, Ps. Vall d'Hebron 119-129 8&z.ausco; Planta, 08035, Barcelona, Spain.
Journal of the neurological sciences (NETHERLANDS) May 1 2000, 176 (1) p65-9, ISSN 0022-510X Journal Code: JBJ
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Hashimoto's encephalopathy is a severe and rather infrequent clinical condition initially described in patients suffering from chronic lymphocytic **thyroiditis**. Its origin is still controversial but it can be agreed to have an **autoimmune** etiology. In fact, its most characteristic finding is the high titre of antithyroid antibodies, especially antimicrosomal. We describe three cases of Hashimoto's encephalopathy and establish a relationship between the clinical status, the antithyroid antibody levels and its response to corticosteroid treatment. There was an excellent response to corticosteroid treatment in all three cases. Interestingly, one case was associated with Graves' disease. Given this, and after the **review** of the literature, we believe that the term 'encephalopathy associated to autoimmune thyroid disease' could be more appropriate to define this entity. Finally, we suggest that autoimmune thyroid encephalopathy must be suspected in the face of unaccounted acute or subacute encephalopathy with high levels of antithyroid antibodies.
Record Date Created: 20000821

7/7/12 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

05588202 88224947 PMID: 3286231
Autoantibodies to the thyrotropin receptor.
Rees Smith B; McLachlan SM; Furmaniak J
Endocrine Immunology Unit, University of Wales College of Medicine, Cardiff, United Kingdom.
Endocrine reviews (UNITED STATES) Feb 1988, 9 (1) p106-21, ISSN 0163-769X Journal Code: EIK
Languages: ENGLISH
Document type: Journal Article; Review; Review, Academic
Record type: Completed
This **review** considers recent developments in our understanding of

the properties of TRAb, particularly measurement of the antibodies and their sites of action and synthesis. Two new assay methods have allowed considerable improvements in the sensitivity, specificity, precision, and ease of measuring TRAb. In particular: 1) receptor assays based on inhibition of receptor-purified labeled TSH binding to detergent-solubilized TSH receptors and 2) bioassays based on stimulation of cAMP release from monolayer cultures of isolated thyroid cells. Detailed studies with the two assays indicate that TSH receptor antibodies nearly always act as TSH agonists in patients with a history of Graves' hyperthyroidism. Studies in areas of dietary iodine sufficiency suggest that measurement of the antibodies at various stages in the course of treating Graves' disease can be of value in predicting the outcome of therapy. However, in areas of iodine deficiency, difficulties in the ability of patients' thyroid tissue to recover from the effects of antithyroid drugs may **prevent** the receptor **antibodies** from causing a relapse of thyrotoxicosis. Consequently, the predictive value of receptor antibody measurements would be expected to be lower in these geographical areas. Although patients with a history of Graves' hyperthyroidism nearly always have TRAb which act as TSH agonists, about 20% of patients with frank hypothyroidism due to autoimmune destruction of the thyroid have TRAb which act as TSH antagonists (**blocking antibodies**). There is some evidence that these **blocking antibodies** can cause hypothyroidism particularly in the neonate. With regard to the site of synthesis of TRAb, there is now direct evidence that they are synthesized by thyroid lymphocytes, particularly the lymphocytes in close proximity to thyroid follicular cells. This is consistent with the well established effects of antithyroid treatment (drugs, radioiodine, or surgery) on TRAb levels in addition to their effects on thyroid hormone synthesis. Recent studies using affinity labeling with 125I-labeled TSH have enabled elucidation of the structure of the TSH receptor. TSH receptors in human, porcine, and guinea pig thyroid tissue have a two-chain structure in which the TSH binding site is formed on the outside surface of the cell membrane by a water-soluble A subunit (Mr approximately 50 K). The A subunit is linked by a disulfide bridge and weak noncovalent bonds to the amphiphilic B subunit (Mr approximately 30 K). This subunit, which penetrates the lipid bilayer, probably forms the site for interaction of the receptor with the regulatory subunits of adenylate cyclase.(ABSTRACT TRUNCATED AT 400 WORDS) (139 Refs.)

Record Date Created: 19880628

7/7/13 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

05381739 89090256 PMID: 2642770

Recent advances in the understanding of humoral and cellular mechanisms implicated in thyroid autoimmune disorders.

Mariotti S; Chiovato L; Vitti P; Marcocci C; Fenzi GF; Del Prete GF; Tiri A; Romagnani S; Ricci M; Pinchera A

Cattedra di Endocrinologia, University of Pisa, Italy.

Clinical immunology and immunopathology (UNITED STATES) Jan 1989, 50 (1 Pt 2) pS73-84, ISSN 0090-1229 Journal Code: DEA

Languages: ENGLISH

Document type: Journal Article; Review; Review, Tutorial

Record type: Completed

In this **review** new data are reported indicating that the thyroid microsomal-microvillar antigen can be identified with thyroid peroxidase (TPO). This concept derives from binding studies of monoclonal and polyclonal microsomal antibodies to TPO purified by affinity chromatography or obtained by recombinant DNA technology. Furthermore, immunofluorescence studies performed on cultured thyroid cells have shown the presence of a TPO-related antigen on the surface of the cells. The expression of the TPO antigen is modulated by TSH through the cAMP pathway. The functional activities of TSH receptor autoantibodies have also been characterized.

From these studies the following conclusions can be drawn: (i) TSH receptor antibodies possess multiple biological activities, interfering or mimicking TSH actions; (ii) a good correlation is observed between stimulation of adenylate cyclase and of iodide uptake by Graves' IgG. In these IgG preparations, adenylate cyclase- and growth-stimulating activities cannot be separated; (iii) **antibodies blocking** the TSH-dependent AC are present in patients with autoimmune hypothyroidism; (iv) a mixture of stimulating and **blocking antibodies** may coexist in the same patient, whose clinical status may result from the sum of the biological activities of these antibodies. Finally, new data are reported on the identification and characterization of T cell clones infiltrating the thyroid tissue of subjects with thyroid autoimmune disorders. The majority of these clones were CD8+ cytolytic T cells with natural killer activity. These latter data may be of importance in the mechanisms of thyroid damage observed in Hashimoto's glands. (63 Refs.)
Record Date Created: 19890214

7/7/14 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

04414158 82010001 PMID: 7024497
Childhood thyromegaly: recent developments.
Reiter EO; Root AW; Rettig K; Vargas A
Journal of pediatrics (UNITED STATES) Oct 1981, 99 (4) p507-18,
ISSN 0022-3476 Journal Code: JLZ
Languages: ENGLISH
Document type: Journal Article; Review
Record type: Completed
Evaluation of a child with goiter includes historical **review**, physical examination, and measurement of serum concentrations of PBI, T4 and T3RU, TSH, and titers of antithyroglobulin and antithyroid microsomal antibodies. If there are no indications for more intensive evaluation such as history of cervical irradiation, a palpable abnormality of the thyroid gland or unusual laboratory findings (e.g., a significant PBI-thyroxine iodine discrepancy in the absence of a positive antithyroid **antibody** titer), a trial of TSH-**suppressive** therapy with thyroxine is undertake, even if the cause of thyromegaly has not been identified. If thyroid size diminishes in the ensuing six to 12 months, treatment is maintained for approximately two years and then discontinued. If the goiter recurs, or if there is impaired thyroid function, treatment is resumed. Periodically, antithyroid antibody titers and indices of thyroid function are determined. If the goiter does not diminish after a reasonable trial of suppressive therapy with adequate amounts of thyroxine (i.e., those quantities which will inhibit TRH-induced secretion of TSH), subtotal thyroidectomy is recommended to be certain that an underlying neoplasm has not been overlooked. A biopsy of the thyroid is not performed routinely in such children prior to operative therapy. Almost invariably, examination of the surgical specimen reveals CLT. Postoperatively, suppressive doses of thyroxine are maintained indefinitely. Inasmuch as thyroxine suppression of TSH secretion is essential in the management of patients with thyroid neoplasms, a limited medical trial, as described, does not place the patient at undue risk. (81 Refs.)
Record Date Created: 19811122

7/7/15 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

135370280 CA: 135(26)370280b JOURNAL
Hypothyroidism in Hashimoto's thyroiditis
AUTHOR(S): Takasu, Nobuyuki
LOCATION: Second Department of Internal Medicine, Ryukyu University,

Japan,
JOURNAL: Annu. Rev. Naibunpi, Taisha DATE: 2000 PAGES: 220-226 CODEN: ARNTC7 LANGUAGE: Japanese PUBLISHER: Chugai Igakusha
SECTION:
CA215000 Immunochemistry
IDENTIFIERS: review hypothyroidism Hashimoto thyroiditis antibody mutation
DESCRIPTORS:
Hypothyroidism... Mutation...
antibodies and genetic mutations in development of hypothyroidism in Hashimoto's thyroiditis
Antibodies...
autoantibodies, TSAb (thyroid-stimulating antibody); antibodies and genetic mutations in development of hypothyroidism in Hashimoto's thyroiditis
Antibodies...
autoantibodies, TSBAb (thyroid-stimulating blocking antibody); antibodies and genetic mutations in development of hypothyroidism in Hashimoto's thyroiditis
Thyroid gland,disease...
autoimmune thyroiditis; antibodies and genetic mutations in development of hypothyroidism in Hashimoto's thyroiditis
Thyroglobulin... Thyrotropin receptors...
genetic mutations in
Transport proteins...
Na+/I- symporter; genetic mutations in
?

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      Set  Items  Description
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Cost is in DialUnits
? b 410

       10mar02 12:50:09 User208760 Session D2019.1
            $0.35    0.099 DialUnits File1
     $0.35  Estimated cost File1
     $0.35  Estimated cost this search
     $0.35  Estimated total session cost   0.099 DialUnits

File 410:Chronolog(R)  1981-2002/Jan
       (c) 2002 The Dialog Corporation

      Set  Items  Description
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HILIGHT set on as ''
HILIGHT set on as ''
? begin (oophoritis or thyroiditis) and (gp39 or cd40L or cd40(w)ligand)

>>>"(" is invalid in a filelist.
? begin 5,73,155,399

       10mar02 12:50:59 User208760 Session D2019.2
            $0.00    0.143 DialUnits File410
     $0.00  Estimated cost File410
     $0.05  TYMNET
     $0.05  Estimated cost this search
     $0.40  Estimated total session cost   0.242 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R)  1969-2002/Mar W1
         (c) 2002 BIOSIS
  File  73:EMBASE  1974-2002/Mar W1
         (c) 2002 Elsevier Science B.V.
*File  73: For information about Explode feature please
see Help News73.
  File 155:MEDLINE(R)  1966-2002/Mar W1
  File 399:CA SEARCH(R)  1967-2002/UD=13610
         (c) 2002 AMERICAN CHEMICAL SOCIETY
*File 399: Use is subject to the terms of your user/customer agreement.
RANK charge added; see HELP RATES 399.

      Set  Items  Description
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? s (oophoritis or thyroiditis) and (gp39 or cd40L or cd40(w)ligand)

            852  OOPHORITIS
          21817  THYROIDITIS
            534  GP39
           3559  CD40L
          13812  CD40
         303673  LIGAND
           6073  CD40(W)LIGAND
     S1      35  (OOPHORITIS OR THYROIDITIS) AND (GP39 OR CD40L OR
                 CD40(W)LIGAND)
? rd s1

...completed examining records
     S2      19  RD S1 (unique items)
? t s2/3/all
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2/3/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13040002 BIOSIS NO.: 200100247151
IL-12 prevents tolerance induction with mouse thyroglobulin by priming pathogenic T cells in experimental autoimmune **thyroiditis**: Role of IFN-gamma and the costimulatory molecules **CD40L** and CD28.
AUTHOR: Zhang Wei; Flynn Jeffrey C; Kong Yi-chi M(a)
AUTHOR ADDRESS: (a)Department of Immunology and Microbiology, Wayne State University School of Medicine, 540 E. Canfield Avenue, Detroit, MI, 48201 **USA
JOURNAL: Cellular Immunology 208 (1):p52-61 February 25, 2001
MEDIUM: print
ISSN: 0008-8749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

2/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12945605 BIOSIS NO.: 200100152754
Characteristics of inflammatory cells in spontaneous autoimmune **thyroiditis** of NOD.H-2h4 mice.
AUTHOR: Yu Shiguang; Medling Brad; Yagita Hideo; Braley-Mullen Helen(a)
AUTHOR ADDRESS: (a)Division of Immunology, Department of Medicine, University of Missouri, M450 Med. Sciences, Columbia, MO, 65212: mullenh@health.missouri.edu**USA
JOURNAL: Journal of Autoimmunity 16 (1):p37-46 Feb., 2001
MEDIUM: print
ISSN: 0896-8411
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

2/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12831914 BIOSIS NO.: 200100039063
Characteristics of inflammatory cells in spontaneous autoimmune **thyroiditis** (SAT) of NOD H2h4 mice.
AUTHOR: Yu S(a); Sharp G C(a); Braley-Mullen H(a)
AUTHOR ADDRESS: (a)Dept. of Med., VA Research Service, U. of Missouri, Columbia, MO, 65212**USA
JOURNAL: FASEB Journal 14 (6):pA996 April 20, 2000
MEDIUM: print
CONFERENCE/MEETING: Joint Annual Meeting of the American Association of Immunologists and the Clinical Immunology Society Seattle, Washington, USA May 12-16, 2000
ISSN: 0892-6638
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English

2/3/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11940545 BIOSIS NO.: 199900186654
CD40L is necessary for the priming of effector cells for lymphocytic and granulomatous experimental autoimmune **thyroiditis**.
AUTHOR: Peterson Karin E; Braley-Mullen Helen(a)
AUTHOR ADDRESS: (a)Division of Immunology and Rheumatology, Department of Medicine, University of Missouri, M450 Me**USA
JOURNAL: Journal of Autoimmunity 12 (1):p1-12 Feb., 1999
ISSN: 0896-8411
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

2/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11577105 BIOSIS NO.: 199800357801
Reduction of atherosclerosis in mice by inhibition of CD40 signalling.
AUTHOR: Mach Francois; Schoenbeck Uwe; Sukhova Galina K; Atkinson Elizabeth ; Libby Peter(a)
AUTHOR ADDRESS: (a)Vascular Med. Atherosclerosis Unit, Cardiovascular Div., Dep. Med., Brigham and Women's Hosp., H**USA
JOURNAL: Nature (London) 394 (6689):p200-203 July 9, 1998
ISSN: 0028-0836
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

2/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10851592 BIOSIS NO.: 199799472737
Suppression of murine **thyroiditis** via blockade of the CD40-**CD40L** interaction.
AUTHOR: Carayanniotis G(a); Masters S R; Noelle R J
AUTHOR ADDRESS: (a)Fac. Med., Health Sci. Cent., 300 Prince Philip Dr., St. John's, Newfoundland A1B 3V6**Canada
JOURNAL: Immunology 90 (3):p421-426 1997
ISSN: 0019-2805
RECORD TYPE: Abstract
LANGUAGE: English

2/3/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10337000 BIOSIS NO.: 199698791918
The relative contribution of the CD28 and **gp39** costimulatory pathways in the clonal expansion and pathogenic acquisition of self-reactive T cells.
AUTHOR: Griggs Nathan D(a); Agersborg Sally S; Noelle Randolf J; Ledbetter Jeffrey A; Linsley Peter S; Tung Kenneth S K
AUTHOR ADDRESS: (a)Box 214, Health Sci. Cent., Dep. Pathology, Univ. Virginia, Charlottsville, VA 22908**USA
JOURNAL: Journal of Experimental Medicine 183 (3):p801-810 1996
ISSN: 0022-1007

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

2/3/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09885688 BIOSIS NO.: 199598340606
Contribution of CD28/CTLA4/B7 and gp39/CD40 costimulation pathways in clonal expansion and functional acquisition of self reactive T cells.
AUTHOR: Griggs Nathan(a); Agersborg Sally; Noelle Randolf; Ledbetter Jeffrey; Linsley Peter; Tung Kenneth
AUTHOR ADDRESS: (a)Dep. Pathol., Univ. Virginia, Charlottesville, VA 22908 **USA
JOURNAL: Journal of Cellular Biochemistry Supplement 0 (21A):p141 1995
CONFERENCE/MEETING: Keystone Symposium on Control and Manipulation of the Immune Response Taos, New Mexico, USA March 16-22, 1995
ISSN: 0733-1959
RECORD TYPE: Citation
LANGUAGE: English

2/3/9 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

07814727 EMBASE No: 1999305192
CD40 expression in human thyroid tissue: Evidence for involvement of multiple cell types in autoimmune and neoplastic diseases
Smith T.J.; Sciaky D.; Phipps R.P.; Jennings T.A.
Dr. T.J. Smith, Div. of Molec./Cellular Med. (A-175), Albany Medical College, 47 New Scotland Avenue, Albany, NY 12208 United States
Thyroid (THYROID) (United States) 1999, 9/8 (749-735)
CODEN: THYRE ISSN: 1050-7256
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 33

2/3/10 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

06609169 EMBASE No: 1996273942
CD40 and its ligand
Clark L.B.; Foy T.M.; Noelle R.J.
Biochemistry Graduate Program, Dartmouth Medical School,Lebanon, NH 03756 United States
Advances in Immunology (ADV. IMMUNOL.) (United States) 1996, 63/- (43-78)
CODEN: ADIMA ISSN: 0065-2776
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

2/3/11 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

136052724 CA: 136(4)52724k PATENT
Methods for regulating a cell-mediated immune response by blocking lymphocytic signals and by blocking LFA-1 mediated adhesion

INVENTOR(AUTHOR): Townsend, Robert M.; Todderud, Charles Gordon; Peach, Robert J.
LOCATION: USA
ASSIGNEE: Bristol-Myers Squibb Company
PATENT: PCT International ; WO 200195928 A2 DATE: 20011220
APPLICATION: WO 2001US18619 (20010608) *US PV210671 (20000609)
PAGES: 75 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-038/17A; A61K-039/395B; A61P-037/00B; C07K-014/705B; C07K-016/28B; A61K-039/395B; A61K-038/17B DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/12 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

135370653 CA: 135(26)370653a PATENT
Compositions and methods for achieving immune suppression
INVENTOR(AUTHOR): Strom, Terry B.; Maslinski, Wlodzimierz; Zheng, Xin Xiao; Kim, Yon Su; Lacraz, Sylvie Ferrari
LOCATION: USA
ASSIGNEE: Beth Israel Deaconess Medical Center, Inc.
PATENT: PCT International ; WO 200187330 A2 DATE: 20011122
APPLICATION: WO 2001US15578 (20010514) *US PV203801 (20000512)
PAGES: 42 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-038/20A; A61K-038/17B; A61K-039/395B; A61P-037/06B; C07K-014/54B; C07K-014/705B; A61K-038/20B; A61K-038/17B DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/13 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

134365711 CA: 134(26)365711t PATENT
Compositions and methods for treating autoimmune diseases and transplant rejections
INVENTOR(AUTHOR): Chu, Keting; Wang, Changyu
LOCATION: USA
ASSIGNEE: Chiron Corporation
PATENT: PCT International ; WO 200134649 A2 DATE: 20010517
APPLICATION: WO 2000US30739 (20001108) *US PV164503 (19991109)
PAGES: 49 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C07K-016/00A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE;

TR; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/14 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

133088219 CA: 133(7)88219b PATENT
Use of CD40 engagement to alter T cell receptor usage
INVENTOR(AUTHOR): Newell, Martha K.; Wagner, David; Newell, Evan
LOCATION: USA
ASSIGNEE: University of Vermont and State Agricultural College
PATENT: PCT International ; WO 200039283 A1 DATE: 20000706
APPLICATION: WO 99US30930 (19991222) *US PV114106 (19981229)
PAGES: 50 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-005/02A; C12N-005/06B; C12N-005/08B DESIGNATED COUNTRIES: AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CU; CZ; DE; DK; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA; UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/15 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

131143419 CA: 131(11)143419c DISSERTATION
The role of secondary signaling in experimental autoimmune thyroiditis
AUTHOR(S): Peterson, Karin Elizabeth
LOCATION: Univ. of Missouri, Columbia, MO, USA
DATE: 1998 PAGES: 218 pp. CODEN: DABBBA LANGUAGE: English CITATION: Diss. Abstr. Int., B 1999, 59(8), 4004 AVAIL: UMI, Order No. DA9904865

2/3/16 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

130223295 CA: 130(17)223295d PATENT
Preparation of imidazoquinoxaline protein tyrosine kinase inhibitors
INVENTOR(AUTHOR): Barrish, Joel C.; Chen, Ping; Das, Jagabandhu; Iwanowicz, Edwin J.; Norris, Derek J.; Padmanabha, Ramesh; Roberge, Jacques Y.; Schieven, Gary L.
LOCATION: USA
ASSIGNEE: Bristol-Myers Squibb Company
PATENT: PCT International ; WO 9909845 A1 DATE: 19990304
APPLICATION: WO 98US16027 (19980803) *US 56770 (19970825) *US 69159 (19971209)
PAGES: 315 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A31K-031/54A; A31K-031/495B; C07D-403/02B; C07D-413/14B DESIGNATED COUNTRIES: AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CU; CZ; DE; DK; EE; ES; FI; GB; GE; GH; GM; HU; ID; IL; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA; UG; UZ; VN; YU; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

2/3/17 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

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130094477 CA: 130(8)94477p PATENT
CD154 blockade therapy for autoimmune diseases
INVENTOR(AUTHOR): Thomas, David W.
LOCATION: USA
ASSIGNEE: Biogen, Inc.
PATENT: PCT International ; WO 9900143 A1 DATE: 19990107
APPLICATION: WO 98US13284 (19980626) *US 51072 (19970627) *US 51481 (19970701) *US 51483 (19970701) *US 51484 (19970701)
PAGES: 33 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A
DESIGNATED COUNTRIES: AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CU; CZ; DE; DK; EE; ES; FI; GB; GE; GH; GM; GW; HU; ID; IL; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA; UG; US; US; US; US; UZ; VN; YU; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH ; GM; KE; LS; MW; SD; SZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE; SN; TD; TG

2/3/18 (Item 8 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

126130597 CA: 126(10)130597f PATENT
Treatment of T-cell mediated autoimmune disorders with antagonists of T-cell receptors mediating contact-dependent helper functions, and anti-gp39 antibody
INVENTOR(AUTHOR): Noelle, Randolph J.; Claassen, Eric
LOCATION: USA
ASSIGNEE: Trustees of Dartmouth College; Nederlandse Organisatie Voor Toegepast-Natuurwetenschappelijk Onderzoek TNO
PATENT: PCT International ; WO 9640246 A1 DATE: 19961219
APPLICATION: WO 96US9137 (19960606) *US 481735 (19950607)
PAGES: 22 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A
DESIGNATED COUNTRIES: AL; AM; AT; AU; AZ; BB; BG; BR; BY; CA; CH; CN; CZ; DE; DK; EE; ES; FI; GB; GE; HU; IL; IS; JP; KE; KG; KP; KR; KZ; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG
DESIGNATED REGIONAL: KE; LS; MW; SD; SZ; UG; AT; BE; CH; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN

2/3/19 (Item 9 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

126017804 CA: 126(2)17804h PATENT
Human antibodies derived from immunized xenomice
INVENTOR(AUTHOR): Kucherlapati, Raju; Jakobovits, Aya; Klapholz, Sue; Brenner, Daniel G.; Capon, Daniel J.
LOCATION: USA
ASSIGNEE: Cell Genesys, Inc.
PATENT: PCT International ; WO 9634096 A1 DATE: 19961031
APPLICATION: WO 95US5500 (19950428)
PAGES: 64 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/00A
DESIGNATED COUNTRIES: AU; CA; FI; HU; JP; KR; NO; NZ
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE
? s (oophoritis or thyroiditis) (20n) (autoimmun?) and review?

852 OOPHORITIS
21817 THYROIDITIS
183049 AUTOIMMUN?

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          10274  (OOPHORITIS OR THYROIDITIS)(20N)AUTOIMMUN?
        3084432  REVIEW?
     S3     588  (OOPHORITIS OR THYROIDITIS) (20N) (AUTOIMMUN?) AND
                 REVIEW?
? s s3 and py=1993

            588  S3
        1866180  PY=1993
     S4      17  S3 AND PY=1993
? rd s4

...completed examining records
     S5      13  RD S4 (unique items)
? t s5/7/all
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Set	Items	Description
S1	7	AU="NOELLE RANDOLPH J"
S2	4	(24(W)31 OR 89(W)76)(30N)(ANTIBOD? OR HYBRIDOMA?) AND (GP39 OR CD40L OR CD40(W)LIGAND)
S3	15	(DIABETES OR THYROIDITIS OR OOPHORITIS)(30N)(ANTIBOD? OR HYBRIDOMA?) AND (GP39 OR CD40L OR CD40(W)LIGAND)
S4	13	(AUTOIMMUN?)(30N)(TREAT? OR THERAP? OR PREVENT? OR INHIBIT? OR SUPPRESS?)(40N)(GP39 OR CD40L OR CD40(W)LIGAND)
S5	101	(AUTOIMMUN?)(30N)(TREAT? OR THERAP? OR PREVENT? OR INHIBIT? OR SUPPRESS?) AND (GP39 OR CD40L OR CD40(W)LIGAND)

ULL TEXT: 2591 lines
? s (autoimmun?)(30n)(treat? or therap? or prevent? or inhibit? or suppress?)(40n)(gp39 or cd40L or cd40(w)ligand)

Processing

	7193	AUTOIMMUN?
	634738	TREAT?
	103540	THERAP?
	1307977	PREVENT?
	297771	INHIBIT?
	148833	SUPPRESS?
	51	GP39
	63	CD40L
	325	CD40
	26963	LIGAND
	129	CD40(W)LIGAND
S4	13	(AUTOIMMUN?)(30N)(TREAT? OR THERAP? OR PREVENT? OR INHIBIT? OR SUPPRESS?)(40N)(GP39 OR CD40L OR CD40(W)LIGAND)

? t s4/3/all

4/3/1 (Item 1 from file: 654)
DIALOG(R)File 654:US Pat.Full.
(c) format only 2000 The Dialog Corp. All rts. reserv.

03117128
Utility
CD40 ANTIGEN ANTIBODY COMPLEX

PATENT NO.: 6,056,959
ISSUED: May 02, 2000 (20000502)
INVENTOR(s): de Boer, Mark, Beverwyk, NL (Netherlands)
Conroy, Leah B, Pacifica, CA (California), US (United States of America)
ASSIGNEE(s): Chiron Corporation, (A U.S. Company or Corporation), Emeryville, CA (California), US (United States of America) [Assignee Code(s): 11661]
APPL. NO.: 8-463,893
FILED: June 05, 1995 (19950605)

CROSS-REFERENCE TO RELATED APPLICATIONS

This application is a division of U.S. Ser. No. 08-070,158, filed May 28, 1993, now U.S. Pat. No. 5,677,165, which is a continuation in part of U.S. Ser. No. 07-910,222, filed Jul. 09, 1992, now U.S. Pat. No. 5,397,703.
FULL TEXT: 1106 lines

4/3/2 (Item 2 from file: 654)
DIALOG(R)File 654:US Pat.Full.
(c) format only 2000 The Dialog Corp. All rts. reserv.

03110270
Utility
ANTIBODIES AGAINST HUMAN CD40

PATENT NO.: 6,051,228
ISSUED: April 18, 2000 (20000418)
INVENTOR(s): Aruffo, Alejandro A., Belle Mead, NJ (New Jersey), US (United

States of America)
Hollenbaugh, Diane, Newtown, PA (Pennsylvania), US (United States of America)
Siadak, Anthony W., Seattle, WA (Washington), US (United States of America)
Berry, Karen K., Princeton, NJ (New Jersey), US (United States of America)
Harris, Linda, Seattle, WA (Washington), US (United States of America)
Thorne, Barbara A., Issaquah, WA (Washington), US (United States of America)
Bajorath, Jurgen, Lynnwood, WA (Washington), US (United States of America)
ASSIGNEE(s): Bristol-Myers Squibb Co , (A U.S. Company or Corporation), New York, NY (New York), US (United States of America)
[Assignee Code(s): 22921]
APPL. NO.: 9-26,291
FILED: February 19, 1998 (19980219)
FULL TEXT: 2068 lines

4/3/3 (Item 3 from file: 654)
DIALOG(R)File 654:US Pat.Full.

03054655
Utility
HUMANIZED ANTIBODIES TO HUMAN GP39, COMPOSITIONS CONTAINING THEREOF

PATENT NO.: 6,001,358
ISSUED: December 14, 1999 (19991214)
INVENTOR(s): Black, Amelia, Cardiff, CA (California), US (United States of America)
Hanna, Nabil, Olivenhian, CA (California), US (United States of America)
Padlan, Eduardo A., Kensington, MD (Maryland), US (United States of America)
Newman, Roland A., San Diego, CA (California), US (United States of America)
ASSIGNEE(s): Idec Pharmaceuticals Corporation, (A U.S. Company or Corporation), San Diego, CA (California), US (United States of America)
[Assignee Code(s): 40498]
APPL. NO.: 8-554,840
FILED: November 07, 1995 (19951107)
FULL TEXT: 2838 lines

4/3/4 (Item 4 from file: 654)
DIALOG(R)File 654:US Pat.Full.

03028899
Utility
53BP2 COMPLEXES

PATENT NO.: 5,977,311
ISSUED: November 02, 1999 (19991102)
INVENTOR(s): Nandabalan, Krishnan, Guilford, CT (Connecticut), US (United States of America)
Yang, Meijia, East Lyme, CT (Connecticut), US (United States of America)
Schulz, Vincent Peter, Madison, CT (Connecticut), US (United States of America)
ASSIGNEE(s): CuraGen Corporation, (A U.S. Company or Corporation), New Haven, CT (Connecticut), US (United States of America)

[Assignee Code(s): 48737]
APPL. NO.: 8-935,450
FILED: September 23, 1997 (19970923)

This invention was made with United States Government support under award number 70NANB5H1066 awarded by the National Institute of Standards and Technology. The United States Government has certain rights in the invention.

FULL TEXT: 5262 lines

4/3/5 (Item 5 from file: 654)
DIALOG(R)File 654:US Pat.Full.

03012757
Utility
COMPOSITIONS COMPRISING A PEPTIDE INHIBITOR OF NUCLEAR PROTEIN TRANSLOCATION AND AN IMMUNOSUPPRESSANT AND METHODS OF USE THEREOF

PATENT NO.: 5,962,415
ISSUED: October 05, 1999 (19991005)
INVENTOR(s): Nadler, Steven G., Princeton, NJ (New Jersey), US (United States of America)
ASSIGNEE(s): Bristol-Myers Squibb Co , (A U.S. Company or Corporation), New York, NY (New York), US (United States of America)
[Assignee Code(s): 22921]
APPL. NO.: 9-72,429
FILED: May 04, 1998 (19980504)

CROSS-REFERENCE TO RELATED APPLICATION

This application is a continuation-in-part of U.S. Ser. No. 08-928,958 filed Sep. 12, 1997, now U.S Pat. 5,877,282, which is related to provisional patent application Ser. No. 60-026,978, filed Sept. 20, 1996, from which priority is claimed under 35 USC 1 19(e)(1). Both of the above-referenced applications are incorporated herein by reference in their entirety.

FULL TEXT: 1555 lines

4/3/6 (Item 6 from file: 654)
DIALOG(R)File 654:US Pat.Full.

02994342
Utility
FUSION PROTEINS COMPRISING GP39 AND CD8

PATENT NO.: 5,945,513
ISSUED: August 31, 1999 (19990831)
INVENTOR(s): Aruffo, Alejandro, Edmonds, WA (Washington), US (United States of America)
Hollenbaugh, Diane, Seattle, WA (Washington), US (United States of America)
Ledbetter, Jeffrey A., Seattle, WA (Washington), US (United States of America)
ASSIGNEE(s): Bristol-Myers Squibb, (A U.S. Company or Corporation), Seattle, WA (Washington), US (United States of America)
APPL. NO.: 8-690,096
FILED: July 30, 1996 (19960730)

This is a division of application Ser. No. 07-940,605, filed Sep. 4, 1992 now issued as U.S. Pat. No. 5,540,926 on Jul. 30, 1996.

FULL TEXT: 1395 lines

4/3/7 (Item 7 from file: 654)
DIALOG(R)File 654:US Pat.Full.

02972983
Utility
SOLUBLE LYMPHOTOXIN-.BETA. RECEPTORS AND ANTI-LYMPHOTOXIN RECEPTOR AND LIGAND ANTIBODIES AS THERAPEUTIC AGENTS FOR THE TREATMENT OF IMMUNOLOGICAL DISEASE

PATENT NO.: 5,925,351
ISSUED: July 20, 1999 (19990720)
INVENTOR(s): Browning, Jeffrey L., Brookline, MA (Massachusettes), US (United States of America)
Benjamin, Christopher D., Beverly, MA (Massachusettes), US (United States of America)
Hochman, Paula S., Brookline, MA (Massachusettes), US (United States of America)
ASSIGNEE(s): Biogen, Inc , (A U.S. Company or Corporation), Cambridge, MA (Massachusetts), US (United States of America)
[Assignee Code(s): 21695]
APPL. NO.: 8-505,606
FILED: July 21, 1995 (19950721)
FULL TEXT: 1791 lines

4/3/8 (Item 8 from file: 654)
DIALOG(R)File 654:US Pat.Full.

02919180
Utility
PEPTIDE INHIBITORS OF NUCLEAR PROTEIN TRANSLOCATION HAVING NUCLEAR LOCALIZATION SEQUENCES AND METHODS OF USE THEREOF

PATENT NO.: 5,877,282
ISSUED: March 02, 1999 (19990302)
INVENTOR(s): Nadler, Steven G., Princeton, NJ (New Jersey), US (United States of America)
Cleaveland, Jeffrey S., Seattle, WA (Washington), US (United States of America)
Blake, James, Seattle, WA (Washington), US (United States of America)
Haffar, Omar K., Seattle, WA (Washington), US (United States of America)
ASSIGNEE(s): Bristol-Myers Squibb Company, (A U.S. Company or Corporation), New York, NY (New York), US (United States of America)
[Assignee Code(s): 22921]
APPL. NO.: 8-928,958
FILED: September 12, 1997 (19970912)
FULL TEXT: 1404 lines

4/3/9 (Item 9 from file: 654)
DIALOG(R)File 654:US Pat.Full.

02918854
Utility
MONOCLONAL ANTIBODIES SPECIFIC FOR DIFFERENT EPITOPES OF HUMAN GP39 AND METHODS FOR THEIR USE IN DIAGNOSIS AND THERAPY

PATENT NO.: 5,876,950
ISSUED: March 02, 1999 (19990302)
INVENTOR(s): Siadak, Anthony W., Seattle, WA (Washington), US (United States of America)
Hollenbaugh, Diane L., Seattle, WA (Washington), US (United States of America)
Gilliland, Lisa K., Bellevue, WA (Washington), US (United States of America)
Gordon, Marcia L., Seattle, WA (Washington), US (United States of America)
Bajorath, Jurgen, Lynnwood, WA (Washington), US (United States of America)
Aruffo, Alejandro A., Edmonds, WA (Washington), US (United States of America)
ASSIGNEE(s): Bristol-Myers Squibb Company, (A U.S. Company or Corporation), New York, NY (New York), US (United States of America) [Assignee Code(s): 22921]
APPL. NO.: 8-379,057
FILED: January 26, 1995 (19950126)
FULL TEXT: 3714 lines

4/3/10 (Item 10 from file: 654)
DIALOG(R)File 654:US Pat.Full.

02915748
Utility
HUMANIZED ANTI-CD40 MONOCLONAL ANTIBODIES AND FRAGMENTS CAPABLE OF BLOCKING B CELL PROLIFERATION
[Antibody which is not agonist of B-cells; treatment of systemic lupus erythematosus, allergies, autoimmune diseases]

PATENT NO.: 5,874,082
ISSUED: February 23, 1999 (19990223)
INVENTOR(s): de Boer, Mark, Heemskerk, NL (Netherlands)
ASSIGNEE(s): Chiron Corporation, (A U.S. Company or Corporation), Emeryville, CA (California), US (United States of America) [Assignee Code(s): 11661]
APPL. NO.: 8-606,293
FILED: February 23, 1996 (19960223)

CROSS-REFERENCE TO RELATED APPLICATIONS

This application is a continuation-in-part of U.S. Ser. No. 08-070,158, filed May 28, 1993 U.S. Pat. No. 5,677,165 now, which is a continuation-in-part of U.S. Ser. No. 07-910,222 filed Jul. 9, 1992, now U.S. Pat. No. 5,397,703, the disclosures of which are hereby incorporated by reference.

FULL TEXT: 1657 lines

4/3/11 (Item 11 from file: 654)
DIALOG(R)File 654:US Pat.Full.

02870957
Utility
TREATMENT OF T CELL MEDIATED AUTOIMMUNE DISORDERS
[Administering an antagonist to a receptor on the surface of the T cells]

PATENT NO.: 5,833,987
ISSUED: November 10, 1998 (19981110)
INVENTOR(s): Noelle, Randolph J., Cornish, NH (New Hampshire), US (United States of America)

Claassen, Eric, Pijnacker, NL (Netherlands)
ASSIGNEE(s): Nederlanse Organisatie Voor Teogepastnatuurwetenschappelijk Onderzoek TNO, (A Non-U.S. Company or Corporation), Rijswijk, NL (Netherlands)
Trustees of Dartmouth College, (A U.S. Company or Corporation), Hanover, NH (New Hampshire), US (United States of America)
[Assignee Code(s): 5263; 5682]
APPL. NO.: 8-481,735
FILED: June 07, 1995 (19950607)
FULL TEXT: 584 lines

4/3/12 (Item 12 from file: 654)
DIALOG(R)File 654:US Pat.Full.

02788393
Utility
RECOMBINANT ANTIBODIES FOR HUMAN THERAPY

PATENT NO.: 5,756,096
ISSUED: May 26, 1998 (19980526)
INVENTOR(s): Newman, Roland A., San Diego, CA (California), US (United States of America)
Hanna, Nabil, Olivenhain, CA (California), US (United States of America)
Raab, Ronald W., San Diego, CA (California), US (United States of America)
ASSIGNEE(s): IDEC Pharmaceuticals Corporation, (A U.S. Company or Corporation), San Diego, CA (California), US (United States of America)
[Assignee Code(s): 40498]
APPL. NO.: 8-476,237
FILED: June 07, 1995 (19950607)

FIELD OF THE INVENTION

This application is a continuation-in-part of U.S. Ser. No. 08-379,072, filed Jan. 25, 1995 (U.S. Pat. No. 5,658,570), which is a continuation of U.S. Ser. No. 07-912,292 (abandoned), filed Jul. 10, 1992, which is a continuation-in-part of Newman et al., U.S. patent application Ser. No. 07-856,281, filed Mar. 23, 1992 (abandoned), which is a continuation-in-part of U.S. patent application Ser. No. 07-735,064, filed Jul. 25, 1991 (abandoned), the whole of which, including drawings, are hereby incorporated by reference. This invention relates to recombinant antibodies useful for human therapy, and to methods for production of such antibodies.

FULL TEXT: 1809 lines

4/3/13 (Item 13 from file: 654)
DIALOG(R)File 654:US Pat.Full.

02552628
Utility
SOLUBLE AND ITS USE IN B CELL STIMULATION
[Gp39 protein]

PATENT NO.: 5,540,926
ISSUED: July 30, 1996 (19960730)
INVENTOR(s): Aruffo, Alejandro, Edmonds, WA (Washington), US (United States of America)
Hollenbaugh, Diane, Seattle, WA (Washington), US (United States of America)
Ledbetter, Jeffrey A., Seattle, WA (Washington), US (United

States of America)
ASSIGNEE(s): Bristol-Myers Squibb Company, (A U.S. Company or Corporation), Seattle, WA (Washington), US (United States of America)
[Assignee Code(s): 22921]
APPL. NO.: 7-940,605
FILED: September 04, 1992 (19920904)
FULL TEXT: 1398 lines